In th Specification:

Please insert the following new paragraph beginning at page 1, line 1 (before "Field of Invention"):

Related Application Information

This application claims priority under 35 U.S.C. § 119(e) to United States Provisional Patent Application No. 60/097,640, filed August 24, 1998.

Please amend the paragraph beginning on page 13, line 27 as follows.

Hsp47 was identified as a protein induced in endothelial cells treated with brefeldin A. In addition to Hsp47 (identified as the ~~465~~ 46.5 KDa band in Figure 14A and as p47 in Figure 14B), other proteins are induced by brefeldin A. These include the protein designated p27 in Figure 14B and a faint band above p47 in Figure 14B having a molecular weight of approximately 60-70 KDa. Such additional proteins may also be immunoprotective polypeptides useful in inhibiting the immune response alone or in combination with all the other proteins induced by brefeldin A.

Please amend the paragraph beginning on page 18, line 22 as follows.

The subject compositions including brefeldin, Hsp47-related polypeptides and expressible nucleic acids encoding such immunoprotective polypeptides or combinations thereof may be also employed in vivo, administrating the subject compositions by any convenient means for the treatment of autoimmune diseases or to facilitate organ or cellular (e.g., bone marrow) transplants. In the case of transplantation, the subject compositions may be administered prior to implantation, administration usually beginning not later than about 14 days prior to implantation, there preferably being at least one dosage administered within three days of ~~administration~~ transplantation. The subject compositions may be administered in the

period beginning about 6 h prior to implantation and may be continued on a predetermined schedule thereafter, usually not past 30 days, more usually not past 20 days. However, after implantation, the subject compositions may be administered as needed, depending upon the response of the recipient to the organ or cells. In some situations, the subject compositions may be administered chronically, as long as the implant is present in the host. Other forms of in vivo use include injection of the subject compositions into areas of ~~inflamation~~ inflammation, e.g., joints, ligaments, tendons and the like as well as various organs such as liver.

Please amend the paragraph beginning on page 29 line 3 as follows:

By sequence homology, Hsp47 is a serine protease inhibitor with theoretical specificity for lysine at the active site of incoming serine proteases. Serpins act as "bait" representing cleavable substrates which form a stable instead of a transitory covalent bond with the active center of serine proteases. Those then do not become released any more, thus blocking the active site of the serine protease they reacted with. Thus, serpins inactivate serine proteases in a stoichiometric ratio of 1:1. Despite the sequence relationship, no substrate serine protease has so far been identified for Hsp47. Some authors expect the serpin-domain to be nonfunctional because its "active site" amino acids are modified (Hirayoshi *et al., Mol Cell Biol* 11:4036-4044 (1991)). We mutagenized Hsp47 in truncational analysis across the serpin domain to evaluate whether a highly specific serine protease of the granule contents of CIK like granzyme A, but were not able to demonstrate a function of Hsp47 as an irreversible inhibitor of attacking granzymes in BLT esterase or SDS-PAGE gelshift assays (data not shown). Our data is paralleled by work with purified murine Hsp47 which failed to inhibit major serine proteases in BLT esterase assays in vitro (Davids *et al., Bioorganic Chemistry* 23:437-438 (1995)). In ^{51}Cr-release assays, deletion of the serpin domain led to loss of function of the affected truncation mutants. However, as further deletions of the gene re-established full

function to the smaller truncated Hsp47 mutants, expressing then little more than the H1, A-A2-consensus region discussed below, we assume that the loss of function of the C-terminal ~~kuncation~~ truncation of Hsp47's serpin domain causes a conformational change in the protein which normalizes with further truncation.